Prions and dentistry

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J R Soc Med 2002;**95**:178–181 SECTION OF PATHOLOGY, 2 OCTOBER 2001

The risk of transmission of prions during dental care is not known, although on existing evidence it is likely to be very low. Although prions may be detected in the oral tissues of inoculated laboratory animals, at present there are no published reports of the detection of prions in non-lymphoid tissues of humans with any form of Creutzfeldt–Jakob disease (CJD). The present article reviews current knowledge of the presence of prions in the mouth, discusses the possible transmission of prions via oral tissues and outlines the possible modifications of infection control measures required for the dental health care of patients with prion disease.

PRIONS IN THE DENTAL HEALTH CARE SETTING

Transmission of sporadic CJD by dental treatment was proposed as long as 20 years ago[1,2]. Later, reporting three patients in Japan with sporadic CJD, Arakawa *et al.*[3] suggested that prions might have been acquired as a consequence of dental treatment but did not provide any supporting data. Case–control studies have never revealed any association between dental health care and the development of either sporadic[4] or iatrogenic[5] CJD, and at present there are no data to suggest any clustering of variant CJD (vCJD) about a dental practice.

Prions in oral tissues

Prions have been detected in the oral and dental tissues of animals with experimental scrapie, and the finding of neuronal degeneration with probable prion protein accumulation in the trigeminal ganglia of patients with sporadic CJD points to a possible route of transmission of prion from the brain to the oral tissues (and *vice versa*)[6]. Inoculation of scrapie agent into the peritoneum or dental pulps of hamsters leads to eventual prion infection of the trigeminal ganglion on the side of inoculation[7], the estimated rate of travel along the trigeminal nerve being 1 mm per day.

Prion protein was not detected in the pulpal homogenates of 8 US patients with sporadic CJD[8]; however, intraperitoneal injection of scrapie agent led to infection of the dental pulps of hamsters after about 96 days[7]. Prions were also detected in the gingival tissues of these animals after about 760 days, the concentration of prion being higher in gingival than in pulpal tissue.

Since prion protein of vCJD is present in tonsillar lymphoid tissue[9] it is likely to be present also in lingual tonsil. In addition, the tendency for prion of vCJD to occur at sites outside the central nervous system suggests that it will be present in the trigeminal ganglion—particularly since the prion of bovine spongiform encephalopathy (BSE) can be present in peripheral nerves[10].

Infectivity

There is no definitive evidence that prion disease can be transmitted by oral tissue, although biting is a possible explanation for accidental transmission of scrapie between encaged animals[11]. Gingival scarification with burrs previously used to scarify scrapie-infected mice did not lead to scrapie[1], although intraperitoneal injection of gingival tissue did give rise to astrocytosis of scrapie in mice. Also, gingival exposure to scrapie-infected brain homogenate caused scrapie in recipient mice[12]. Of note, while gingival scarification (e.g. with forceps and scissors) caused transmission to all laboratory animals, disease also developed in 71% of animals gingivally exposed to brain homogenate but not scarified. Early studies suggested that gingival extracts have low infectivity[1,12]—for example, intracerebral inoculation of gingival tissue from scrapie-infected mice only caused scrapie in 3 of 31 mice[12]—but later work revealed substantially greater levels of prion protein in gingival than in pulpal tissue of scrapie-infected hamsters[7].

There is little information on the precise infectivity of prion-infected oral tissues. One study of scrapie-infected hamsters established that the infectivity of pulpal tissue was $5.6 \log LD_{50}$ while that of gingival tissue was $72 \log LD_{50}$. These were lower than the infectivities of trigeminal ganglion and brain tissue.

Likelihood of transmission of prions during dental health care

First a word about experience with nosocomial transmission of non-prion infectious agents during dentistry. Hepatitis B virus was at one time readily transmitted during dental care, but contemporary infection control measures have reduced this risk to almost zero[13].

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Although 6 patients probably acquired HIV as a consequence of care by an HIV-infected dentist in the USA[14], lookback studies of the patients of other HIV-infected dental staff have not disclosed any patients infected with this virus as a result of dental treatment[15]. Dentists may be liable to hepatitis A virus infection, at least as evidenced by HAV seroprevalence studies[16], although they do not seem to be at risk of occupational acquisition of either hepatitis C virus[17] or Transfusion Transmitted Virus[18]. No dental health care worker (DHCW) is believed to have been infected with HIV as a consequence of occupational injury[15].

Clearly, these agents are much more easily activated than prions. Epidemiological evidence offers some reassurance that prions are not likely to be transmitted to DHCWs during dental treatment but the possibility cannot be excluded.

Oral manifestations of prion disease

Oral manifestations of human transmissible spongiform encephalopathies are dysphagia and dysarthria (due to pseudobulbar palsy), and in vCJD patients there may be orofacial dysaesthesia or paraesthesia[19]. Loss of taste and smell has been reported in one patient with vCJD[20].

Possible routes of transmission of prions during dental care

Since prion protein of vCJD is likely to be in perioral lymphoid tissue, and prions of scrapie can be transmitted via pulpal and gingival tissue, we must assume that there is some risk, albeit small, of prion protein transmission during dental care. The most likely means of transmission would be via contaminated dental instruments; thus measures to reduce this risk are essential in the dental surgical care of patients with known prion disease.

GUIDELINES FOR THE DENTAL MANAGEMENT OF PATIENTS WITH PRION DISEASE

Existing guidelines for the clinical management of patients with prion disease do not address dental health care in any detail[21–23], although this subject has been discussed elsewhere[24]. In general the suggested infection control procedures for the dental management of patients with known prion disease are similar to those of all other patients, with certain important modifications. At present oral tissues are considered to be of low infectivity, so persons liable to iatrogenic CJD (i.e. recipients of dura mater, corneal transplants and human pituitary hormones and persons who have undergone neurosurgical procedures) are considered at low risk of prion transmission, hence no additional infection control measures are recommended other than those employed in universal cross-infection control[22].

Instruments must not be reused but discarded appropriately

The current UK guidance is that all health care instruments employed in the treatment of a patient with *known* prion disease should be discarded[21,22]. Single-use instruments are preferred, and these will come into increasing use for all patients as new legislation comes into force.

Dental unit waterlines must not be activated

Dental unit waterlines can become contaminated with prions when the dental handpiece is connected to the waterline. Thus, in view of the present impossibility of inactivating prions, a sensible policy is to avoid the risk of retraction of prions into the waterlines by instead using a coolant provided by syringe.

Dental unit waterlines are a potential source of nosocomial infection. Dental staff have an excess seroprevalence of influenza A and B viruses and respiratory syncytial virus[24], this possibly being due to the generation of aerosols[25] and the development of biofilms within the lines. Dental staff also have an increased seroprevalence of legionella, and titres may correlate with duration of dental practice[26]. Although the frequency of legionella seropositivity does not correlate with rates of clinical disease[27], fatal *Legionella dumoffii* in one dentist may have been due to acquisition of infection from bacteria within the waterline[28]. The precise risk of acquisition by patients or dental health care staff of infection from waterlines is not known, but since two immunosuppressed patients developed nonfatal infection of *Pseudomonas aeruginosa* derived from dental unit waterlines[29] there is clearly a risk.

Biofilms of microorganisms derived from both the water source of the unit and retracted oral fluids develop within 8 hours within waterlines[30]. The rapid biofilm formation reflects the low diameter to surface area of the lines, the ease of adherence of bacteria to the hydrophobic polyurethane or polyvinyl surface, the low rate of flow of water and the frequent long periods of no flow within the line[31,32].

Retraction of oral fluids into dental handpieces and the waterline is common, indeed as much as 800 μL of fluid can pass into the handpiece[33]. Bacteria (e.g. *Ps. aeruginosa*) and viruses (e.g. HIV, hepatitis B, herpes simplex, bacteriophage 174) have been found to be retracted into the waterlines[34]. Flushing even for 10–20 minutes does not remove the biofilm[35–38] (since the pressure at the tubing wall is almost zero[31]) and currently no antiretraction system, filtering mechanism[32], or biocide has been reported to remove biofilms consistently from dental unit waterlines.

Thus, since there is a risk of retraction of prions in oral fluids, it would seem sensible not to activate waterlines when patients with known prion disease require restorative dental care. There is little information on the possible retraction of materials into the *air* lines of dental units.

An independent suction and spittoon other than those of the dental unit should be used

In view of the difficulties of disinfection, the suction system of the dental unit cannot be used; instead a stand-alone suction unit should be used. The reservoir of the suction unit should be disposable. Patients should expectorate into a disposable bowl, not a spittoon, and this should be discarded directly into the clinical waste bin for incineration.

CONCLUSIONS

Epidemiological evidence does not suggest that prion transmission as a consequence of dental health care has occurred, but work in animals has established that the oral tissues can become infected with prions and be a potential source of infection in other animals. There is a need for research to establish the potential susceptibility of oral tissues to infection by prions of vCJD and bovine spongiform encephalopathy, and to determine the exact infectivity of prion-containing oral tissues. At present the dental instruments of patients with known prion disease should be discarded after use. In view of the possible risk of contamination with prions due to retraction of oral fluids it is advisable not to use the waterlines or suction systems of dental units when treating patients with known prion disease.

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